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RESPIRATORY ACTIVITY IN SPEECH

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ABSTRACT. Functionally the subglottal respiratory system behaves for the most part like a piston in a piston chamber driven with a constant force (the sum of thoracic and abdominal muscular forces plus the elastic recoil force). Small, short term, variations in subglottal air pressure occur nevertheless due to (a) short-term variations in the resistance to the exiting airflow, i.e., from glottal and supraglottal articulations and (b) the inertia of the pulmonic system which creates delays in its passive response to such short-term variations in subglottal pressure. This casts doubt on claims of active contribution of the pulmonic system in the implementation of syllables, stress, certain sentence-final fundamental frequency contours, and certain segment types, e.g., aspirated stops.

1. INTRODUCTION

Speech consists primarily of the articulators transforming the dc pressure variations created during respiration into ac pressure variations or sound.[1] Speech is further superimposed almost exclusively on the expiratory phase of respiration[2] although this expiration is often prolonged far beyond what it would be in ordinary respiration. In this paper I will review research and claims on what the pulmonic system does during speech. Since a great many basic reference works cover the anatomy and physiology of the respiratory system in general and in speech

1 The glottalic and velaric speech sounds found in some languages, that is, clicks, ejectives, and implosives, are created out of dc pressure variations created by piston-like action of the tongue and/or the larynx (Catford 1939, Ladefoged 1962a).

2 The exceptions are ingressive speech used in vocal disguise, speaking when out of breath (e.g., when reporting emergencies), and interjections in various cultures, e.g., French "oui" or Scandinavian "ja" or "nej".

(e.g., Zemlin 1968, Luchsinger & Arnold 1965, Hixon 1973, di Cristo 1975, Catford 1977) I will not go into great detail on these matters but will instead focus on the less well resolved issues relating specifically to the involvement of the pulmonic system in speech.

2. ANATOMY

Respiration involves getting oxygen into and carbon dioxide out of the lungs which are two sponge-like lobes having great internal surface area in order to facilitate gas exchange. This is accomplished by varying the volume and thus the pressure in and around the lungs by muscular and passive elastic forces acting on the thoracic cavity. Although more than a dozen muscles can be involved in respiration under certain conditions, including any of the trunk muscles, the primary muscles used are the internal and external intercostals (which connect the ribs), the diaphragm (the thin dome-shaped muscular sheet separating the abdominal cavity and the thoracic cavity) and the various sheet-like muscles covering the abdominal cavity which are, in effect, antagonistic to the diaphragm. Of these, the external intercostals and the diaphragm are primarily associated with inspiration and the internal intercostals and the muscles covering the abdomen, primarily with expiration (although see below).

The lungs plus the thoracic and abdominal cavities also have an elasticity which gives rise to forces which oppose the volume changes induced by muscular action and which, when the muscular action ceases or diminishes, tend to restore the system to a certain resting or state.

The lungs of an adult male can hold maximally about 7 liters of air but have a resting capacity of about 4 liters. The latter volume, the so-called functional residual capacity (FRC), is the volume assumed by the lungs when the external airway (trachea, pharynx, plus mouth or nasal passage) is open and the net volume-changing forces acting on the thoracic cavity are zero.

The difference between the 7 l maximum volume and the 4 l at the FRC is the 3 l that can be taken in during a maximum inspiration; a maximum expiration can reduce the volume by 2 l, leaving 2 l of residual volume, the quantity of air that always remains in the lungs. The 5 l that can be exchanged (3 l under maximum inspiration plus 2 liters under maximum expiration) is called the vital capacity.

3. PHYSIOLOGY

There are two somewhat divergent accounts of the physiological activity of respiration during speech. Weismer (1985) refers to these as the 'classic' and 'contemporary' views. They differ primarily in the role assigned to the abdominal system and in the extent to which the accounts are justified by reference to biomechanical efficiency.

3.1. The 'Classic' Account of Respiration During Speech

According to the classic view, quiet breathing typically involves a respiratory cycle that is initiated by an inspiration consisting of an approximately 500 cc increase in lung volume (over the FRC) accomplished by contraction of external intercostals. Expiration is accomplished by the elastic recoil force which brings the lungs back to the FRC. Deep inspiration will also involve more forceful action of the external intercostals in addition to the diaphragm and associated muscles. Forced (rapid) expiration and deep expiration past the FRC also require the engagement of expiratory muscles (internal intercostals). In controlled expirations, which includes speech, the inspiratory muscles may be active when lung volume is above the FRC in order to oppose the elastic recoil force, that is, to prevent a too rapid decrease in lung volume. Short speech intervals (2 or 3 seconds or less) of low to average loudness may be accomplished entirely on the volume of air taken in during a normal inspiration typical of quiet breathing and, like quiet breathing, not require active involvement of expiratory musculature. Anything longer or louder requires a deviation from normal respiration, that is, either inspiring greater volumes of air and/or expiring volumes of air past the FRC.

3.2. The 'Contemporary' Account of Respiration During Speech

The contemporary view, while acknowledging that there are considerable idiosyncratic variations among speakers, emphasizes the crucial role of the abdominal system in respiration. Most speakers are found to make some use of the abdominal system at least to assist the thoracic system in the controlled expirations during speech and some speakers exploit the abdominal system more than the rib cage to change lung volume (Baglioni 1922, Gray 1936, Hixon, Goldman, & Mead 1973, Hixon, Mead, & Goldman 1976, Weismer 1985). Most speech utterances, especially those in conversational style, are said to be initiated on that level of lung volume which would generate the desired subglottal pressure (P_s) purely by the elastic recoil force (c. 60% of vital capacity). After initiation, however, the expiratory force must be supplemented by muscular action in order to maintain the P_s at this level. Most of the expirations on which speech is superimposed are said to be terminated near the FRC. Weismer offers speculations in terms of bioenergetics for the greater efficiency of this scheme over that depicted in the classic view.

3.3. What is Common to the 'Classic' and 'Contemporary' Accounts

The classic and contemporary accounts of speech respiration agree in attributing to the respiratory physiology the goal of providing a relatively constant P_s (for a given loudness level). They agree furthermore that this is accomplished by providing a nearly constant net force to the thoracic cavity and thus the lungs. They differ only in the specifics of how this is done: whether largely with the rib cage muscles, or assisted by the abdominal system, and in how the muscles interact with the elastic recoil force. From the point of view of the

end-product, then, the respiratory system's function in speech is relatively simple: apply a constant force to the lungs in order to maintain P_s at a range useful for the supraglottal articulators to make sound. The complexity arises in implementing this task, given that the forces applied by separate components of the system, including the back pressure of the air inside the lungs themselves, may change radically during a single expiration.

3.1. The Principal Aerodynamic Respiratory Variables

3.1.1 Volume Velocity. The rate of airflow, the volume velocity, U , (units = cm^3/sec), is a function of both the pressure differential between lungs and atmosphere and the resistance offered to the airflow; resistance in turn is a function of the channel characteristics, primarily the area at its narrowest point (aperture). Theoretically the variations in flow as a function of aperture depend on whether the flow is laminar (smooth) or turbulent. The transition from laminar flow to turbulent flow, again theoretically, is supposed to take place suddenly when a certain combination of factors including the physical properties of the fluid (the warm moist air in the case of speech), the channel characteristics and the particle velocity [3] reach a certain value, the so-called Reynolds number. Practically, however, in non-uniform tubes--which is the case with the respiratory tract--there is turbulent flow at almost any rate of air flow and so one is actually dealing with cases where the volume velocity, U , varies with pressure drop, ΔP , and area of the aperture, a , as in (1).

$$U = c \cdot \Delta P^\alpha \cdot a^\beta \quad (1)$$

where c is a constant and the exponents α and β vary non-linearly as a function of particle velocity (Jaeger & Matthys 1970). In general, though, it is safe to take α as 0.5 and β as 1.

3.1.2. Pressure. Variations in air pressure in the respiratory tract are a function primarily of two factors: pressure varies inversely with the volume of the chamber in question and directly with the mass of air in the chamber. These functions are given in the equations in (2) and (3).

$$\frac{V_1}{V_2} = \frac{P_2}{P_1} \quad \text{or} \quad P_2 = \frac{V_1}{V_2} \cdot P_1 \quad (2)$$

$$\frac{M_1}{M_2} = \frac{P_1}{P_2} \quad \text{or} \quad P_2 = \frac{M_2}{M_1} \cdot P_1 \quad (3)$$

The influence of temperature variations are typically ignored under the assumption that pressure changes are essentially adiabatic.

3 Particle velocity, v , is a function of volume velocity and the cross-dimensional area of the channel, $v = U/a$.

(In addition a complete model would take into consideration inertial and elastic factors as well as compliance, the amount by which the surface of the respiratory tract yields to impinging pressure.)

3.2. Interactions Between Pulmonic and Oral Systems

Most of the issues surrounding respiratory activity during speech seek to determine the contribution of the pulmonic system independent of activity in the upper respiratory tract, that is, by the glottis plus supraglottal articulators (hereafter designated 'oral articulators' for convenience). Difficulties arise due to interactions between the two systems. Variation in the pressure in the lungs (for our purposes, equivalent to P_s) is a function both of the expiratory forces (muscular plus elastic recoil--by equation (2)) and of the resistance to air flow encountered downstream (by equations (1) and (3)). The same can be said of air flow. Variation in lung volume might be considered a definitive way to differentiate between activity initiated by the lungs and that due to oral articulators but even here complications arise. Variation in the rate of lung volume decrement could be a reflection of independent augmentation of the expiratory force or it could be a largely passive reaction to variation in the expiratory volume velocity. Only by sampling multiple parameters would it be possible to differentiate independent pulmonic activity from that dependent on upper tract activity.

To make this clear, consider a few simple cases. Assume, first, a constant muscular force acting to reduce lung volume--a situation that can be imagined as a weight resting on an inflated balloon. If no air leaves the lungs due to the exit aperture being zero, that is, during breath holding after equilibrium is achieved, then the forces inside the lungs exactly oppose the net force compressing the lungs (muscular force plus recoil force). In this static case there is no change in lung volume and lung pressure is at its maximum. If the exit aperture is non-zero, then there is some airflow and the lungs lose some mass of air--the amount determined by equation (1)--and has its pressure momentarily lowered as reflected in equation (3). When the pressure (force) inside the lungs is then momentarily less than the combined muscular and recoil force compressing the lungs, the latter will dominate and the lung volume will decrease. This lung volume decrement will in turn tend to increase lung pressure (by equation (2)). Given the inertia of the system, however, during a continuous venting of the air from the lungs there is some phase lag between the air mass decrement and the lung volume decrement; consequently the pressure in the lungs during expiration is less than that of the combined muscular and recoil force.

Evidence for this latter point is the fact that during intervocalic voiceless stops the pressure sampled just below the glottis (P_s) rises above the level of that for adjacent vowels. See Fig. 1. In such cases the variations in lung pressure and exit volume velocity are a function of "downstream" activity.

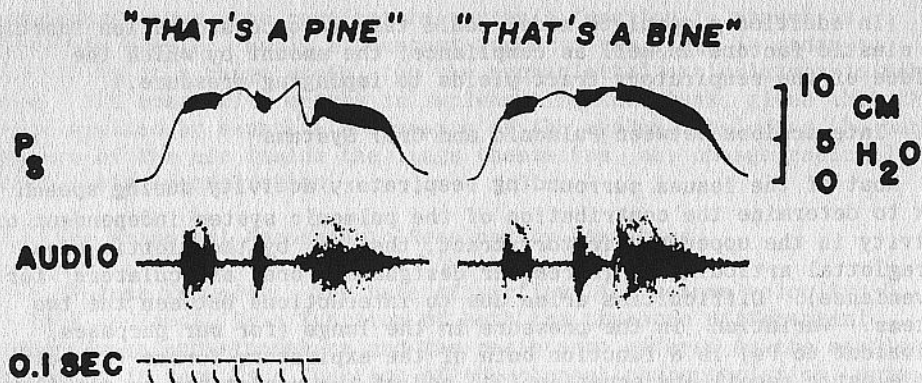


Figure 1. Subglottal pressure and microphone signal sampled during two utterances spoken by an adult male speaker of English.

On the other hand, consider the case of an unvarying exit aperture but a varying muscular force. Here the muscular variations will cause directly proportional variations in lung pressure (by eq. (2)) and in the airflow leaving the lungs (by eq. (1)). In this case variations in lung pressure and exit volume velocity are a function of "upstream" activity.

3.3. Measurement

A variety of physiological respiratory parameters may be sampled during speech.

3.3.1. Electromyography. The activity of various muscles may be sampled using electromyography (EMG) (Stetson 1951, Draper, Ladefoged, & Whitteridge 1959, Mead, Bouhuys, & Proctor 1968, Munro & Adams 1971, van Katwijk 1974). The main difficulty with this method is that since many of the relevant muscles are not near the surface or if they are, are overlaid or adjacent to other muscles, surface electromyographic electrodes offer problems. This leaves needle electrodes which (a) require considerable skill to use and (b) restrict the subject population.

3.3.2. Plethysmography. The variations in the volume of the lungs may be measured relatively easily and non-invasively with the use of a whole body plethysmograph (Mead 1960, Arutjunjan, Granstrom, & Kozhevnikov 1967, Hixon 1972, Ohala 1977a). When a subject is placed inside an airtight container slightly larger than his body and allowed to breathe air from outside the container (e.g., via a close-fitting face mask in the wall of the container), then the variations in the chest or abdominal volume during breathing and speaking will cause proportional changes in the pressure inside the container. Alternatively, one may measure the amount of air passing through a hole in the wall of the container due its displacement by the body volume changes. The former, a pressure plethys-

mograph has better time-resolution but is somewhat more difficult to calibrate (in terms of variations in the volume of the body); the latter, a flow plethysmograph tends to have less time resolution but is easy to calibrate. Other methods of measuring chest volume include belts that wrap around the chest, changes in the length of which reflect changes in the thoracic circumference (e.g., the accordion-like tube which is a familiar part of the polygraph or 'lie detector'; see also Scripture 1902 [1973], pp. 214ff. and Panconcelli-Calzia 1924: 5ff.) and newer devices which transduce chest or abdominal diameter by detecting change in the motion of a sensor placed on one part of the body relative to another reference device (e.g., the sensor detects strength of magnetic field and the reference device emits a magnetic field) (Hixon 1971).

3.3.3. Aerodynamic. Airflow from the mouth and nose and air pressure in the trachea are commonly used in studies addressing issues of the respiratory contribution in speech. These are familiar techniques in instrumental phonetics and will not be reviewed here (see Keller 1971, Ladefoged 1967.) It should be said, however, that most indirect ways of measuring P_s are far inferior to the most commonly used direct method which involves a tracheal puncture. However, as with needle electrodes in EMG, tracheal punctures can only be done by a skilled physician and are unacceptable to many subjects.

4. RESPIRATORY ACTIVITY IN SPEECH

The principal issues concerning the role of the pulmonic system during speech are: what role does it play in the production of stress, syllables, individual speech sounds (phonemes), intonation, including the gradual declination of voice fundamental frequency (F_0) from beginning to end of a breath group (that uttered on a single expiration)?

4.1. Air Under Pressure.

Perhaps the only function of the respiratory system in speech which has never been in dispute is that the lungs provide the air under pressure which, by the action of the oral articulators, is turned into audible pressure variations, i.e., sound. Because the rise and fall of the thorax during speech is quite visible and since kinesthetic sensations relating to expiration are so evident, this has been known from ancient times. Some of the earliest genuinely scientific attempts at speech synthesis, that by Wolfgang von Kempelen (1791) and by Erasmus Darwin (1803, note XI), simulated the function of the pulmonic system quite adequately by means of a bellows. There was some confusion, however, over how the expired air was turned into sound until the matter was cleared up by Willis (1830, 1833).

4.2. Voice Quality

The voice teacher's term "chest voice" as opposed to "head voice", etc. reflects a rather ancient belief that certain modes of phonation, especially as used in singing, emanated from the chest, the forehead,

etc. This, of course, is physiological nonsense (as explicitly acknowledged by Rush 1827:104) although it may have pedagogical, i.e., heuristic, value.

4.3. Loudness

Research by Ladefoged and his colleagues and others have established that, other things being equal (e.g., the shape of the supraglottal configuration), loudness is correlated with Ps (Ladefoged & McKinney 1963, Allen 1971). This by itself does not necessarily mean the pulmonic system is the exclusive cause of voice loudness due to the above-discussed fact that increased Ps can also come about due to increased resistance at the glottis. In fact, there is clear evidence that loud voice is accompanied by a change in the glottal waveform, specifically, one in which the vocal cords are closed for a longer time during each glottal cycle. However, there is also evidence of increased activity by the pulmonic system itself during loud voice (Hixon 1973, Hixon, Goldman, & Mead 1973, Hixon, Mead, & Goldman 1976).

4.4. Syllables

Stetson (1929, 1951 [retrospective ed. 1988]) claimed that individual syllables were produced with ballistic pulmonic pulses which resulted in 'breath pulses' (momentary increases in the pressure and flow rate of expiratory air) which were further shaped (released, checked) by the oral articulators, thus giving rise to the segments or phonemes of speech.[4] He presented a variety of physiological records in support of his claim, including chest wall movements, aerodynamic, and electromyographic data. However, Draper, Ladefoged & Whittridge (1959) and Ladefoged (1962), using more refined EMG methods and a wide variety of speech sample types, showed that Stetson's claims could not be supported. Stetson had evidently been misled by the pressure variations that develop in the lungs due to variations in the resistance encountered by the expiratory airflow due to "downstream" segmental closures and openings.

4 A related but inverse claim regarding syllable production was proposed by James Rush (1827)--inverse in the sense that he seemed to suggest that the respiratory muscles released air in syllable-sized chunks. In his own words: "By our voluntary power over the muscles of respiration, the breath in speech is frugally dealt out to successive syllables, in such small portions as may be requisite for the time and force of each." [107]. In some of his works Henry Sweet also proposed an account of syllables similar to that of Stetson's, e.g., "A syllable is a vowel, either alone or in combination with consonants, uttered with a single impulse of stress" (1891:229). As noted below, he also attributes 'stress' to an action of the respiratory system. Neither of these authors elaborate on their views to the same extent as Stetson nor do they present any supporting instrumental evidence.

4.5. Stress and Other Aspects of Intonation

4.5.1. Introduction. The belief that stress (or the prominence given to individual syllables) is implemented by greater respiratory effort is quite old. I don't know exactly how old, but I have found references to emphasis as 'a mode of sound requiring a greater profusion of breath' in Foster(1763) (and carefully differentiating this from accent which he relates to the pitch of voice and from quantity or the duration of speech sounds). Foster refers to Scaliger (1610) as expressing similar views in his *Diatriba de Europaeorum linguis*. Sweet (1911) declared "Stress is, organically, the result of the force with which the breath is expelled from the lungs; while acoustically it produces the effect of loudness..." Jones (1922) essentially follows Sweet in this view although he acknowledges (p. iv) the work of Coleman (1914) which, in the case of English, emphasizes instead the importance of pitch (F0) changes for the production of stress. Others who thought pitch primary for stress include Mitford (1804), Abas (1925), and Morris (1925), Muyskens (1931), Scott (1939). The importance of pitch variations for the perception of stress (and secondarily, greater duration) has been overwhelmingly supported by modern investigations (Bolinger 1958, Fry 1958) involving a wide variety of languages (including English, Polish, Czech, German, and Dutch; see references in Ohala 1977a). Perhaps the ultimate verification of these findings has been the fact that they have led to highly successful speech synthesis-by-rule (Mattingly 1966, Umeda 1976).

Stetson, while allowing that there might be pitch changes on accented syllables, thought that it would be a secondary effect of the increased expiratory force since

... the heavy stroke of the accent involves the chest pressure and is apt to change the pitch because the laryngeal musculature is often affected by tensions in the other musculatures of speech. [141]

However, he cites no evidence supporting the claim of special sensitivity of the laryngeal muscles to other muscles' contraction and I know of no such evidence that has been presented in the meantime. Nevertheless, it has been known since Müller's research in 1837 (see Mueller 1851:vol. 2, p. 212ff) that increases in Ps can cause slight increases in F0, so the views that greater expiratory force and pitch changes are essential for stress are not logically incompatible.

Stetson presented evidence (records of oral and subglottal air pressure and chest movements) that these allegedly separate 'breath pulses' for each spoken syllable had greater magnitude on stressed syllables.[5]

5 It is interesting to compare Stetson's views with those of Roudet (1910:182ff), published earlier, who was one of the first to study a wide range of aerodynamic parameters in speech (Roudet 1900a,b). Like

Fonagy (1958), studying Hungarian, reported that increased respiratory muscular activity was a more reliable correlate of stress than variations in intensity, F0, or duration.

Ladefoged (1962b, 1967) and his colleagues found a momentary increase in the activity of the expiratory muscles (sampled via EMG needle electrodes) and in Ps during and sometimes immediately before a stressed syllable, whether emphatically stressed or not. He calibrated the effect that variations in Ps would have on both intensity (see above) and F0 (Ladefoged 1963). Although Ps was found to be primarily responsible for the major variations in intensity of voice, the major part of the F0 variations including those on stressed syllables could be attributed to action of the larynx via changes in the tension of the vocal cords.

4.5.2. Recent Controversies. Lieberman (1967) attributed a greater role for the pulmonic system and the variations in Ps which it created than had previously been considered--not only in causing the F0 variations associated with stress but also the utterance-final F0 fall found in declarative statements. The basis for his claim was recordings from three speakers of American English of the acoustic speech signal and Ps during a variety of utterances. He noted that there was generally a close temporal coincidence between a momentary F0 rise on stressed syllables and a momentary increase in Ps and likewise the fall in F0 at the end of declarative sentences and the fall in Ps. (Similar observations had been made previously by others who sampled Ps during speech, e.g., Smith (1944).)

There were numerous problems with Lieberman's claim (see Isshiki 1969, Vanderslice 1970, Ohala 1970, 1977a, 1978, 1982, Ohala & Ladefoged 1970):

1. The inference that Ps was responsible for certain F0 variations in speech rested on the assumption that the laryngeal muscles (which were not sampled in his study) were inactive. There was, however, no basis for this assumption.

2. Controlled calibrations of the effect which independent Ps variations could have on F0 in the modal register (i.e., where care was taken to insure that laryngeal tension was not varying) gave values in

Stetson, he also found variations in either Ps or in oral airflow related to the production of separate syllables. However he more reasonably (from our present viewpoint) attributed these to variations in either glottal or supraglottal resistance. One finds Ps peaks when some closure or constriction is made downstream, i.e., during the production of a consonant. Oddly, Stetson made no reference to Roudet's work in either the first or second edition of *Motor Phonetics* even though both Rousselot (1897-1908) and Scripture (1902), to whom Stetson makes extensive reference, give reviews of Roudet's work.

the range of 2 to 5 Hz/cm H2O (Mueller 1851:vol 2, p. 212ff, Ladefoged 1963, Öhman & Lindqvist 1966)--too low to account for most of the observed F0 variations in speech, given the magnitude of accompanying Ps variations. Öhman and Lindqvist commented, in addition,

...the fo changes during the stressed syllables do not correlate well with the stress-induced ΔP changes either in phase or in amplitude.

Lieberman reported a 16 to 22 Hz/cm H2O influence of Ps on F0 based on the unsupported assumption that vocal cord tension was constant during the samples he obtained. In fact, if his measurements had been done according to the criteria he specified (and any reader can check this since, commendably, Lieberman published all the data from his study), one would obtain values of up to 28 to 33 Hz/cm H2O. These are an order of magnitude higher than values obtained under controlled conditions (then or now--see Titze 1989).

3. Most importantly, (as mentioned earlier) Ps varies not only due to changes in the pulmonic force but also due to changes in the resistance to airflow downstream. There is abundant evidence that changes in F0 and intensity do involve changes in glottal pulse shape, including mean glottal aperture (Roudet 1910: 183, Sonesson 1960, Isshiki 1969, Ishizaka & Flanagan 1972). Indirect evidence of increased glottal resistance during stress syllables (in comparison to unstressed syllables) comes from the occasional (but not consistent) finding that oral airflow is reduced on stressed syllables (Klatt, Stevens, & Mead 1968--see their figure 5--; Broad 1968; this same relation is evident in aerodynamic data obtained by Celia Scully from some of her subjects, personal communication). Atkinson (1973, 1978), whose work Lieberman cites as support for his earlier claims, also fails to consider how Ps is partially dependent on laryngeal state.

Subsequently, there have been several electromyographic studies which have uniformly indicated that F0 variations in speech are implemented by activity of the laryngeal muscles (Ohala & Hirano 1967, Hirano, Ohala, & Vennard 1969, Hirano & Ohala 1969, Fromkin & Ohala 1968, Ohala 1970, 1972, Lieberman, Sawashima, Harris, & Gay 1970, Atkinson 1973, Netsell 1969, 1973, Collier 1975, Maeda 1975, Shipp 1975, Erickson & Atkinson 1975, Kakita & Hiki 1975, Erickson, Baer, & Harris 1983). The muscles involved in raising F0 are the cricothyroid, thyrohyoid, lateral crico-arytenoid, and vocalis; those involved in lowering F0 are the sternohyoid and sternothyroid.

In spite of the array of evidence which fail to support Lieberman's claim that Ps can contribute in any significant way towards the F0 variations in stress and utterance-final fall, Lieberman and Blumstein (1988) still maintain that

[when] stressing a particular word... the primary determinant of fo variation is the subglottal air pressure. [107]

... the "easiest" or "most natural" way of producing a breath-group appears to be the state of minimal [laryngeal] control that results in a terminal falling fo contour... [i.e., due to falling Ps]. [201]

Even though it is clear now that any pulmonic contribution to the F0 changes observed on stressed syllables must be small, this does not rule out the possibility of a pulmonic contribution to other aspects of stress, in particular increases in intensity. Ladefoged (1963, 1967, 1968) citing his own extensive electromyographic investigations of the activity of the expiratory muscles during speech, maintains that there is a momentary increase in expiratory activity during or slightly preceding each stressed syllable, whether emphatically stressed or not. However, Munro & Adams (1971), using needle electrodes to record respiratory activity during speech in four speakers of English, failed to find any 'obvious localised bursts of internal intercostal activity with each stressed syllable.' Van Katwijk (1974) obtained surface electromyographic recordings of the activity of some of the expiratory muscles during speech (of two native speakers of Dutch) and found appreciable increases in their activity only on emphatically stressed syllables, not on conversationally stressed syllables.

It is difficult to evaluate such EMG records by themselves for two reasons. First, there is no simple way of estimating the magnitude of lung volume decrement caused by any bursts of expiratory muscle activity (if found). After all, it is only by decreasing the lung volume at a faster-than-normal rate that these muscles can have any influence on Ps. It is possible that short-term fluctuations in the level of expiratory muscle activity produce only negligible variations in Ps. Second, it is difficult to know why the expiratory muscles might show increased activity near stressed syllables. Is its long-term function to maintain a constant pressure in the lungs or to maintain a constant rate of lung volume decrement, or some combination of the two? If its task is to maintain a steady rate of lung volume decrement, then the momentary increase in expiratory activity on stressed syllables could simply be a compensatory reaction (even in advance) to the increased glottal resistance to airflow expected, which increased resistance would otherwise momentarily slow the rate of lung volume decrease. If this were so, the increased activity of the pulmonic muscles would not, strictly speaking, be an independent feature of stress; it would be dependent upon downstream changes in resistance to airflow.

In order to clarify some of these issues, I obtained records of lung volume during speech.

4.5.3. Plethysmographic Studies. A whole-body pressure plethysmograph was used. It consisted of 1 and 2/3 steel drums welded together with a housing that fitted on top to accommodate the standing subject's head and in one wall of which was face mask through which the subject breathed atmospheric air. The inside volume was approximately 325 l.

Speech samples were obtained from four adult speakers: two male speakers of American English (LB and JJO), one male Swedish speaker, and one female speaker of Hindi. In all cases recordings of the acoustic speech signal were also obtained although these were not of very high quality given the distortions caused by the face mask. Approximate calibrations of the lung volume signal was attempted only in the case of the recording with the Hindi speaker. In the case of one of the English speakers (JJO) and the Hindi speaker, combined oral and nasal airflow was sampled simultaneously and averages of some of the parameters sampled were computed (based on 9 or 10 tokens each) along with the first derivative of the averaged lung volume signal. (Further details may be found in Ohala, Riordan, & Kawasaki 1980 and M. Ohala 1980.)

Figs. 2a,b shows microphone and lung volume curves during single tokens of the sentence 'deem unilluminable real', first in conversational style (a), and then with emphatic stress on the syllable [lum] (b). Figs. 3a-d shows the same parameters during single tokens of 'deem oon real' [dʌm ūn ŋi+], [6] where the blank was filled in with [m, s, h, t], respectively. Fig. 4 shows averaged lung volume and its first derivative (slope) for the Hindi nonsense word [ʔibʰi] (here and with other averaged curves, the vertical dotted line indicates the synchronization point used to form the averages). Figure 5 shows averages of F0, lung volume and the oral plus nasal airflow for the English sentence 'Lure Bill near'. Fig. 6. gives averaged airflow and differentiated lung volume for 5 Hindi nonsense words with [p, b, m, p^h, b^h] in intervocalic position. Fig. 7. exhibits averaged lung volume and airflow for the sentences 'You drew Will near' (on the left) and 'you drew pill near' (on the right).

Based on such data, the following observations were made:

1. There are relatively large rapid decreases in lung volume during moments of high oral airflow, e.g., during aspiration, [h], and fricatives. See Figs. 3, 4, 6, 7. These presumably represent a passive collapse of the lungs due to the rapid flow of air out of the lungs and the consequent decrease in lung pressure. This contrasts with the case of sonorant consonants (nasals, laterals, etc.) where the rate of lung volume decrement is the same as that for surrounding vowels. See Figs. 3, 5, 6, 7.

2. There are also moments of less than normal lung volume decrement during periods of reduced airflow, e.g., as occurs during the closure phase of stops or affricates. See Figs. 2, 4, 5, 6, 7.

3. There are momentary greater-than-normal decreases in lung volume during emphatically stressed syllables. See portion delimited by broken

4. The use primarily of high vowels in the speech samples was necessitated by the face mask.

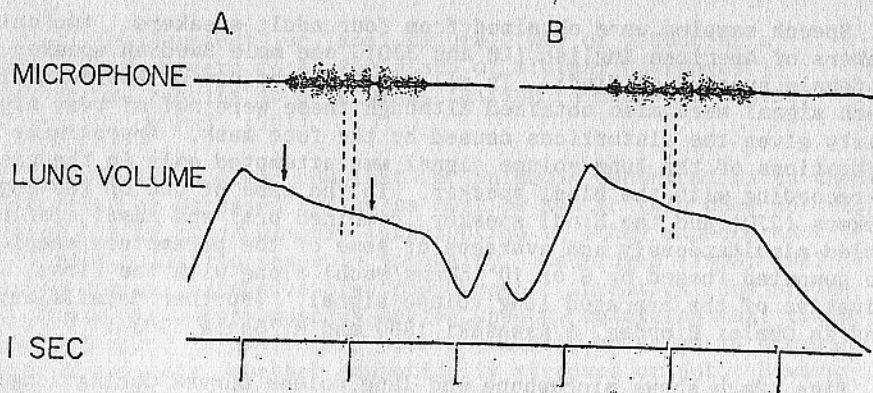


Figure 2. Single tokens of 'deem unilluminable real', in normal conversational style (left) and with heavy stress on the syllable 'lum' (right). Top: microphone signal; bottom: lung volume. Dotted lines delimit the stressed syllable 'lum' in both cases. Arrows mark the momentary plateaus in the lung volume parameter due to the stop closures of the [d] and [b].

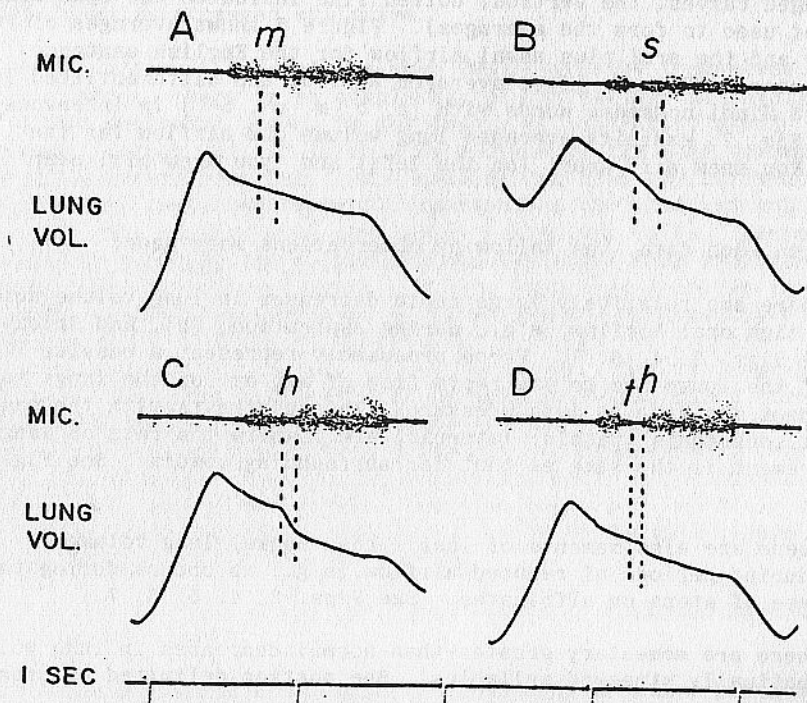


Figure 3. Individual tokens of the frame sentence 'deem ___oon real' where the blank was filled by the consonants [m s h th]; dotted lines delimit the consonants' onsets and offsets. Top: microphone; bottom: lung volume.

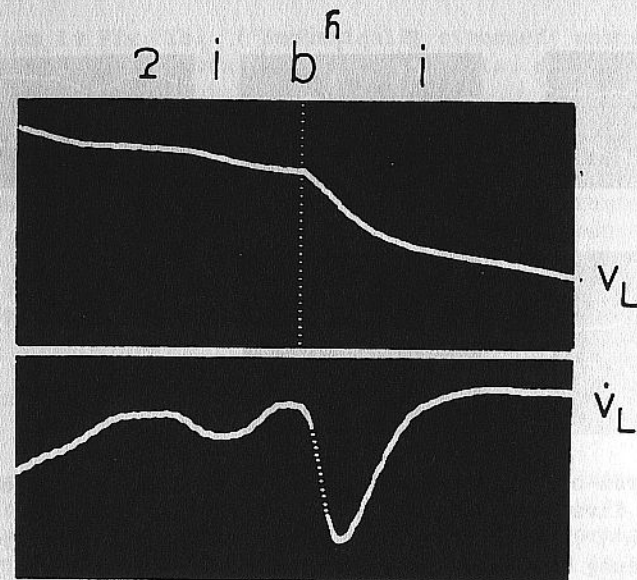


Figure 4. Averaged traces of lung volume (top) and differentiated lung volume (bottom) during the Hindi nonsense utterance [ʔibʰi]. The dotted line marks the consonant release and the point of synchronization for the averages.

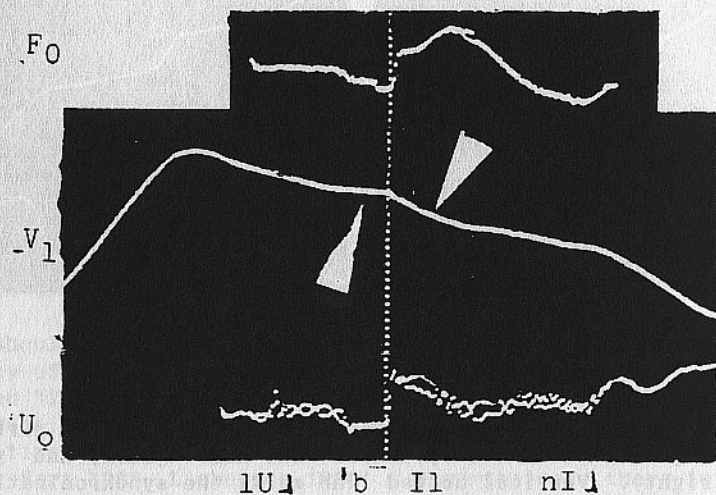


Figure 5. Averaged traces of fundamental frequency (top), lung volume (middle), and oral plus nasal airflow (bottom) during the utterance 'lure Bill near'. Dotted line marks the consonant release and the point of synchronization for the averages.

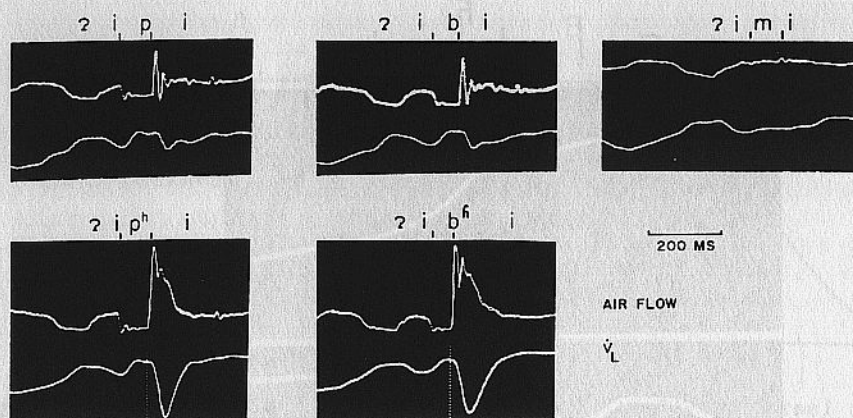


Figure 6. Records of airflow (top) and differentiated lung volume (bottom) during five Hindi utterances: [ʔipi], [ʔibi], [ʔimi], [ʔipʰi], [ʔibʰi]. Oral airflow represents a single token; the differentiated lung volume is the average of 9 or 10 tokens of each utterance. The dotted vertical line marks the consonant release and the synchronization point for the averages.

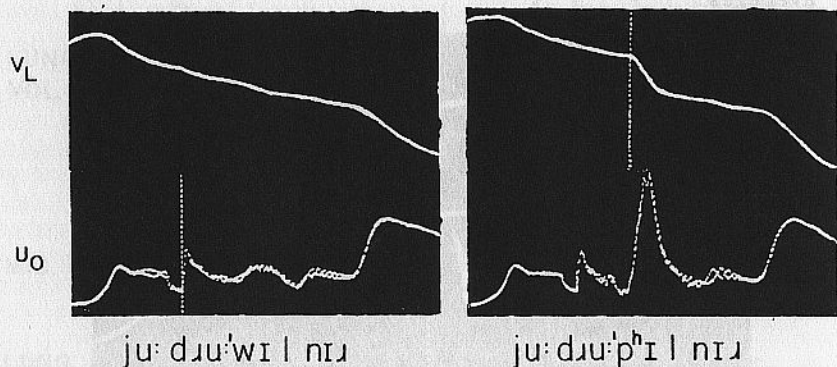


Figure 7. Averages of lung volume (top) and oral plus nasal airflow (bottom) during the utterances 'you drew Will near' (left) and 'you drew pill near' (right). Vertical dotted line marks the synchronization point used to form the averages (left: the release of the [d] in 'drew'; right: the release of the [p] in 'pill').

vertical lines in Fig. 2b. ('Emphatically stressed' means the kind of very forceful emphasis given in heated arguments, dynamic oratory, and the like.)

4. There need not be any obvious change in the rate of lung volume decrement on non-emphatically stressed syllables, i.e., during normal conversational speech. This is easiest to see in sentences consisting entirely of sonorants since the segments themselves induce no perturbation in the rate of airflow out of the lungs. See portion delimited by broken vertical lines in Fig. 2a which shows no change in rate of decrement on the stressed vowel.

5. In general, there is a close short-term (and inverse) relationship between lung volume decrement and the volume of airflow exiting through the mouth and nose. See Figs. 5, 6 and 7.

I interpret these results as showing that the primary function of the pulmonic system during speech is simply to produce P_s that is reasonably constant and above some minimal level. Short-term passive variations in lung volume decrement occur in reaction to variations in lung pressure which in turn are reactions to changing downstream resistance to airflow created by oral articulations. Active short-term variations in lung volume decrement are probably limited to the production of variations in the loudness of speech: lesser decrement for less intense speech, greater decrement for very loud speech.

4.6. F0 Declination

It has been widely observed that the F0 of voice gets progressively lower from beginning to end of a sentence (Pike 1945: 77, Maeda 1974, Pierrehumbert 1979). In African tone languages this results in tones at the end of a sentence being lower in F0 than those at the beginning. Sometimes a high tone may be even lower at the end than a low tone at the beginning (Silverstein 1976). Among Africanists this is called 'down-drift'; the phenomenon as it applies (potentially) to any language is called 'declination' (Cohen & 't Hart 1965). Declination may be absent in questions where instead the failure of F0 to fall helps to differentiate them from declarative utterances.

It should be mentioned that doubt has been expressed about the generality of declination, especially in spontaneous speech--as opposed to planned "laboratory" speech (Lieberman, Katz, Jongman, Zimmerman, Miller 1985a; but see the ensuing exchange between these authors and their critics: Repp 1985, Lieberman, Katz, Jongman, Zimmerman, Miller 1985b, 't Hart 1986, Lieberman 1986). Nevertheless, there is good evidence that listeners expect declination in sentences; for two accented syllables in the same sentence to sound equally prominent the second should have a lower F0 than the first (Pierrehumbert 1979; however, cf. Cohen, Collier & 't Hart 1982).

Assuming that declination does exist in at least some utterances and

is wide-spread among languages (Weitzman 1970, Silverstein 1976), there has been considerable speculation on the physiological mechanisms which cause it. In particular, given its apparent pervasiveness, many have sought to identify automatic, i.e., mechanical and non-purposive, physiological causes: something which changes subtly and progressively during an utterance and which could influence F0. Naturally, attention was focused on the respiratory system. Maeda (1974) suggested that the larynx progressively lowers during a single speech expiration ("breath group") due to its muscular linkage to the sternum which should lower as the lung volume decreases. Given this and the fact that there is often a correlation between larynx height and F0 [7], this movement could cause a gradual lowering of F0. However, the strength of this posited mechanical linkage between the sternum and larynx is called into question by the observation of Ewan (1979:36) that the larynx normally moves upward, not downward, during expiration.

Collier (1975), while acknowledging that the major linguistically-significant F0 variations in speech (e.g., those signalling stress, question-vs-statement) are implemented by the laryngeal muscles, suggested that the F0 variation in declination was caused by the gradually falling Ps which in turn was undoubtedly connected to the progressive increase in expiratory effort required to maintain a sufficiently high Ps as the elastic recoil force decreases when the lung volume approaches the FRC and then opposes expiration as it passes below the FRC. Consistent with this view, he pointed out, were two other facts: (a) there seemed to be no indication in EMG records from the laryngeal muscles during speech that they show any change that can be correlated with the F0 decline and (b) that the rate of F0 change with respect to Ps change during declination was in the range cited above (2-5 Hz/cm H20) which is compatible with independent Ps-caused F0 variation.

This issue cannot be regarded as settled. It is true that more systematic EMG studies addressing the mechanisms underlying declination are needed but there already exist some suggestive data on the issue. In fact, some of the figures published in Collier (1974; an expanded version of Collier 1975) seem to show graded muscle activity during a period of declination; see Fig. 8 ('Contour 15' in the original). In this figure (as well as in 'Contour 11' of the original) the F0 in the latter portion of the utterance is high but gradually falling at about the same rate as in other instances of declination. The Ps contour does not match the F0 trend but the cricothyroid activity does. Maeda (1975) has also presented EMG data which could be interpreted as showing progressive lessening of cricothyroid activity and increase in sternohyoid activity during speech showing the typical F0 declination.

Collier's conclusion that the covariation between Ps and F0 during declination implies that it is Ps causing the F0 change is subject to the

7 The reasons for this correlation are not clear but see Ohala (1972, 1977a, 1977b, 1982). Ohala & Eukel (1987).

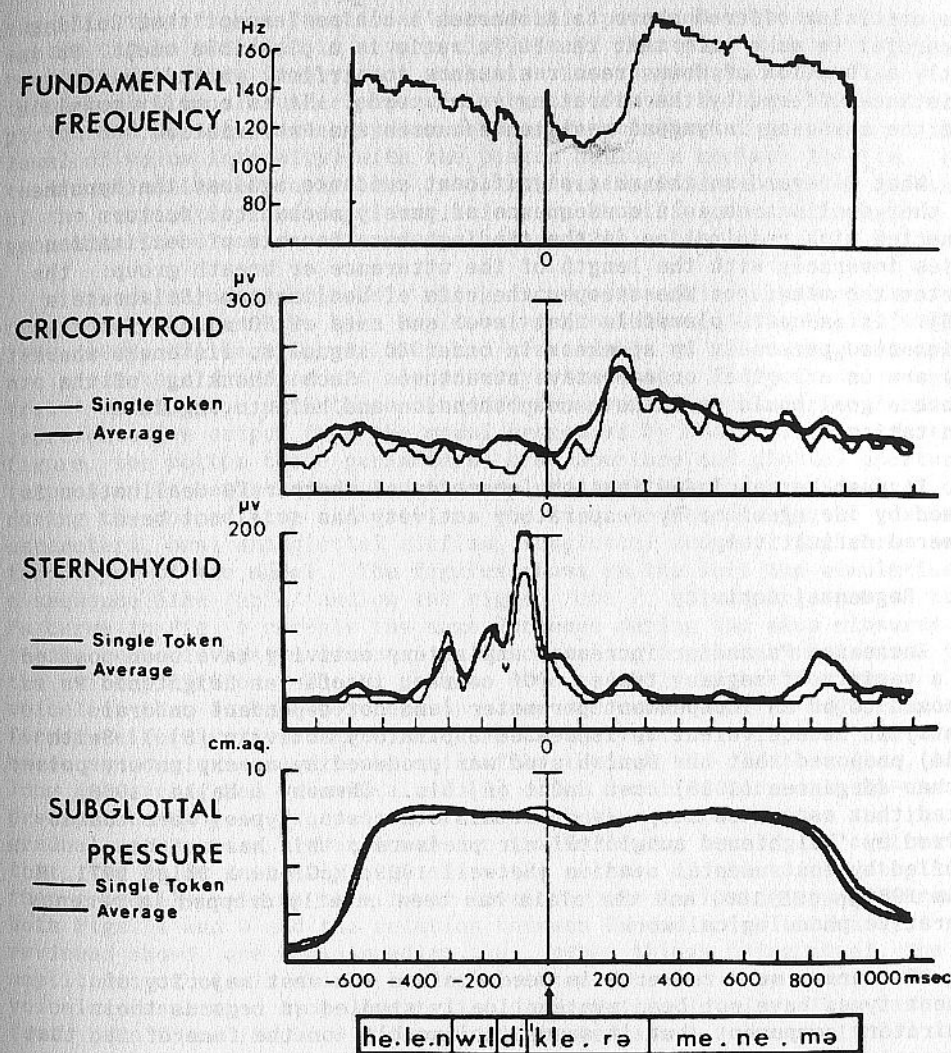


Figure 8. Sampled parameters relevant to F0 control in speech (from Collier 1974). Top to bottom: F0, cricothyroid EMG, sternohyoid EMG, subglottal pressure. The utterance is a Dutch sentence spoken by a native speaker.

same criticism offered above to Lieberman's claims (except that Collier is careful to make sure that the F_0/P_s ratio is a plausible one): P_s is partly a function of downstream resistance to airflow, including the resistance offered by the vibrating vocal cords. It is equally possible that the changing laryngeal resistance causes the P_s variation.

What I regard as the most significant evidence against the hypothesis that declination is a consequence of purely mechanical factors connected with respiration is the finding that the rate of declination varies inversely with the length of the utterance or breath group: the shorter the utterance the steeper the rate of declination (Silverstein 1976). It is quite plausible that level and rate of F_0 modulation are implemented purposely by speakers in order to signal to listeners where they are in a logical or narrative structure. Such "chunking" of the speech signal could facilitate comprehension and help to regulate turn-taking.

It must be concluded that the question of whether F_0 declination is caused by laryngeal or by respiratory activity has still not been answered definitively.

4.7. Segmental Activity

Increased P_s and/or increased expiratory activity have been posited for a variety of segment types. (Of course, insofar as heightened P_s is supposed to be an independent parameter (and not dependent on oral events) it is equivalent to increased expiratory activity [8].) Smith (1944) proposed that the Danish *stød* was produced by an expiratory pulse; Fischer-Jørgensen (1988) cast doubt on this. Chomsky & Halle (1968) stated that aspirated stops (and certain other stop types) were characterized by 'heightened subglottal air pressure'; this has not been verified by instrumental studies (Netsell 1969, McGlone & Shipp 1971, M. Ohala 1983pp.155-160) and the claim has been quietly dropped in recent generative phonological work.

Of course, more research is needed since the vast majority of segment types have not been systematically studied as regards their respiratory component, but it seems implausible, on the face of it, that the massive respiratory system with its very high inertia would show active modulations at a rate corresponding to that at which segments succeed each other in running speech.

5. MODELLING THE RESPIRATORY SYSTEM IN SPEECH

Although further research on the respiratory system in speech is clearly needed, especially multiparametric ones, the preceding review

8 Heightened P_s as a dependent, and therefore non-distinctive, feature of voiceless (as opposed to voiced) stops was demonstrated by Ohala (1975); see below.

suggests that from a functional aerodynamic point of view the respiratory system can be modelled in a very simple way. That is, considering the respiratory system simply as a mechanism for supplying the oral articulators with air under pressure, the system can be modelled as a piston and piston-chamber system driven by a constant force (for a given level of voice intensity) with the piston having a certain inertia. Due to a combination of variations in downstream resistance and the inertia of the respiratory 'piston', variations in P_s will occur, although generally these would not exceed ± 2 cm H₂O.

Speech aerodynamic models which incorporate this scheme of the respiratory system have been implemented (Rothenberg 1968, Ohala 1975, 1976, 1983, Müller & Brown 1980) and are capable of generating aerodynamic parameters (airflow and air pressure at various sites in the vocal tract) which agree with those obtained from real speech. Fig. 9 shows representative output from the model presented by Ohala (1975). In the figure, the bottom three parameters, oral aperture and glottal aperture (expressed as resistances) and the oral volume (which increases by 2 cm during the voiced stop closure) are the input to the model; the remaining parameters, oral and glottal airflow, subglottal and oral pressure, are the output of the model. The figures shows on the left the simulation of a sequence like /ap^ha/ and, on the right, /aba/. A comparison with the P_s curve in Fig. 1 reveals the same increase during the stop closures due to the pulmonic system momentarily encountering a greater resistance to the exiting airflow. The P_s peak is also slightly higher for the voiceless than for the voiced stop since the resistance is higher in the former (effectively infinite) whereas in the latter it is less and must be so as long as some air may still flow through the glottis, i.e., as long as voicing can be maintained.[9] Also modelled correctly is the precipitous drop in P_s upon the release of the aspirated stop and its gradual return to a higher level for the following vowel. In contrast to that, the P_s is initially high after the release of the voiced stop and then gradually returns to a lower level for the following vowel. From both Figs. 1 and 9 and the relation between P_s and voice intensity, reviewed above, one would predict that, other things being equal, the amplitudes of vowels should be higher after a voiced stop than after a voiceless aspirated stop, due to the different P_s levels. This has, in fact, been found (House & Fairbanks 1953, Lehiste & Peterson 1959).

Further evidence of the primacy of glottal and oral articulations in implementing the linguistic units of speech is given by the success of articulatory synthesis which concentrates on articulatory movements from the glottis and above (Scully 1987).

9 This does not constitute confirmation of the Chomsky & Halle claim that aspirated stops differ from unaspirated by the feature [+ Heightened Subglottal Pressure]. The higher P_s here would be true of any voiceless obstruent (vis-a-vis a cognate voiced one), aspirated or not, and, moreover, is a feature dependent on the specified glottal and supraglottal events.

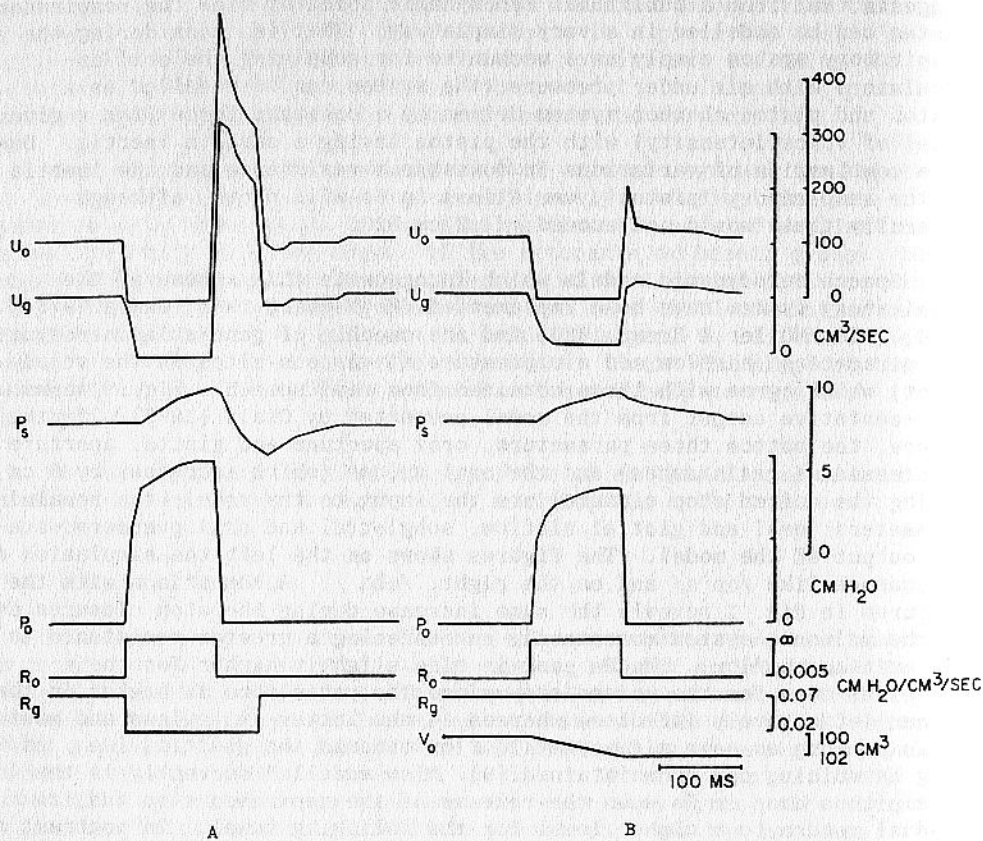


Figure 9. Input and output of the model of speech aerodynamics presented by Ohala (1974). Input consists of the lowest three parameters: (from bottom to top) oral volume, glottal resistance, oral resistance; output, top four parameters: oral pressure, subglottal pressure, glottal airflow, oral airflow. A: an utterance like [ap^ha]; B: one like [aba].

No doubt we can expect future models of speech respiratory mechanics to go beyond simple functional models and to parcel out the expiratory force to elastic recoil, chest and abdominal muscles and to incorporate sufficient physiological and anatomical detail to be able to calculate the biomechanical efficiencies of different modes of expiration, as discussed by Weismer (1985).

6. CONCLUSION

The picture that emerges from the evidence reviewed is that the respiratory system (the pulmonic cavity and the forces which change its volume and the air pressure inside) is functionally quite simple. For a given intensity of voice, the force applied to the lungs is essentially constant; observed short-term variations in the rate of lung volume decrement and in subglottal air pressure are thus caused by two factors, (a) variations in the resistance to airflow offered downstream, i.e., by the oral articulations and (b) the inertia of the respiratory system which prevents it from recovering immediately from the momentary variations in internal lung pressure. Claims that independent action of the respiratory system underlies the production of syllables, stress, certain sentence-final FO contours, FO declination, and certain segment types are called into question.

7. ACKNOWLEDGEMENTS

I thank the participants of the NATO ASI, especially Celia Scully and Ray Kent, for helpful comments on an earlier draft of this paper. Some of the research reported here was conducted in collaboration with Minoru Hirano, Haruko Kawasaki, Manjari Ohala, and Carol Riordan and was supported by the National Science Foundation.

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